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Wernicke's Encephalopathy Complicating Hypermesis Gravidarum: a Rare Case Report

Nawfal Caidi1, Hanaa Lazhar[2]*, Aziz Slaoui[2], Aicha Kharbach[2], Aziz Baydada[2] and Rajae Tachinante[1]

[1]*Department of Anesthesia and Critical Care, University Mohammed V, Morocco*

[2]*Department of Gynaecology Obstetrics and Endoscopy, University Mohammed V, Morocco*

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***Corresponding author:** Hanaa Lazhar, Department of Gynaecology-Obstetrics and Endoscopy, Maternity Souissi, University Mohammed V, Rabat, Morocco

Abstract

Introduction: Wernicke's Encephalopathy (WE) is an uncommon neurological disorder due to thiamine deficiency. Its clinical findings are variable steering its high incidence postmortem. Seldom, thiamine deficiency is present in a clinical triad consisting of cognitive disorder, ocular abnormalities and gait ataxia. Most commonly, the clinical picture is not specific leading to late or misdiagnosis. In pregnant woman with Hyperemesis Gravidarum, incoercible vomiting and poor intake paves the path to thiamine deficiency by depleting B1 storage affecting cerebral energy homeostasis. Ergo, HG leads to neurological complications affecting maternal mortality and morbidity.

Case Report: We hereby present the uncommon case of WE complicate HG in 18 weeks' gestation primigravidae. Diagnosis relied on clinical findings as the clinical picture was typical: confusion, disorientation and memory impairment besides static and kinetic cerebellar syndrome and ophthalmological abnormalities such as unequal pupils and horizontal nystagmus. Biological findings were out of normal range as it showed electrolytes imbalance, dehydration and elevated liver and pancreatic enzymes. Imaging investigations were normal. Treatment was based on intravenous thiamine supplementation and vomiting management.

Conclusion: We are a preventable neurological disorder. Prompt treatment and correct dosage in light of the appropriate guidelines enlighten the prognosis and may even lead to remission. Whereas late or non-diagnosis leads to dramatic complications such as Korsakoff's syndrome. This article aims to raise clinician's suspicion of WE and emphasizes the importance of preventive thiamine supplementation during pregnancies and prophylactic intravenous thiamine administration in pregnancy with Hyperemesis Gravidarum.

Keywords: Wernicke's encephalopathy; Hyperemesis gravidarum; Thiamine; Pregnancy

Abbreviations: WE: Wernicke's Encephalopathy, HG: Hyperemesis Gravidarum, MRI: Magnetic Resonance Imaging, CT: Computed Tomography, MICU: Maternal Intensive Care Unit, ASAT: Aspartate Amino Transferase, ALAT: Alanine Amino Transferase

Introduction

Wernicke's encephalopathy (WE) due to causes other than alcoholism is a rare and often misdiagnosed neurological disorder. It can occur as a complication of many pathologies where the common denominator is malnutrition and malabsorption. Vomiting and nausea during pregnancy is found in more than 80% of pregnant women. However incoercible vomiting lasting for more than 3 weeks is the definition of Hyperemesis Gravidarum and may lead to the onset of WE. Wernicke's encephalopathy in a patient with hyperemesis gravidarum was first reported by Sheehan in 1939. We, also, hereby present the uncommon case of a WE are complicating a primigravidae at 18 weeks' gestation

Case Report

We hereby present the uncommon case of 19 years old primigravida at 12 weeks 'gestation transferred to the maternal intensive care unit (MICU) for a consciousness disorder. The patient used to have regular menses, lasting 5 days. Her history dates back to the second month of pregnancy where she spent a week in the gynecology ward for incoercible vomiting refractory to oral symptomatic treatment. The patient had been discharged with a prescription including an antiemetic (Metoclopramid 10mg/12h) and multivitamin supplementation (Pyridoxin 50mg, Thiamin 250mg and Cyanocobalamin 250mg) to carry on till her next ap-

pointment in the out-patient unit. Meanwhile, the persistence of vomiting and the onset of confusion and mild neurocognitive symptoms such as irritability and apathy, led her husband to take her to the emergency gynecology department and then was immediately transferred to the MICU.

Her physical examination revealed a cachectic, dehydrated, and sub-icteric patient weighing 45Kgs vs 50 2 months ago. Her blood pressure was at 90/50 mmHg and her heart rate was at 125bpm. Her neurological examination showed a confusional state, temporal and spatial disorientation and memory impairment. Besides, it manifests static and kinetic cerebellar syndrome, abolition of osteotendinous reflexes and hypoesthesia mainly in the lower limbs. Her ophthalmological examination exhibited a horizontal Nystagmus, unequal pupils and retinal hemorrhage.

After proper resuscitation, upon the abnormalities in her physical examination and context, WE were suspected and empiric thiamine therapy was administered intravenously before biological and imaging investigations at a dosage of 500mg diluted with normal saline, over 30minutes, three times a day alongside metoclopramid 10mg / 2ml every 8 hours and a Proton-Pump Inhibitor 40mg/J Intravenously. Her complete blood count (CBC) manifested a high rate of hemoglobin, hematocrit and platelets respectively at 16g/dl, 52% and 550000. The rest of her biological analysis objectivized hypokalemia (2.1) a mild hepatic cytolysis (ASAT=140 U/l, ALAT =240)U/l moderate elevation of pancreatic enzymes (Lipasemia at 80U/l) and metabolic acidosis. Lumbar puncture was unremarkable. Imaging tools -Computed Tomography and Magnetic Resonance Imagine- were within normal. Albeit normal imaging investigations and non-specific biological dehydration findings, hyperemesis gravidarum accompanied by the clinical triad of memory impairment, cerebellar signs and ocular symptoms all point to Wernicke encephalopathy. Therefore, We maintained Thiamine intravenous administration. Treatment was continued for the duration of symptoms.

Her stay at the MICU was uneventful. On the 10th day, we noticed the absence of vertical nystagmus and retinal hemorrhage. Her pupils were equal and reactive. However, gait ataxia and memory impairment didn't totally resolve. The patient was left with residual gait disturbance and amnesia. The course of therapy switched to oral Thiamine 100mg every 8 hours on the 20th day post-admission. The patient was discharged after 25 days. Her postoperative care was unremarkable. She is now at 32 weeks gestation. She is still stable with same residual symptoms. Her pregnancy is marked by a small gestational age.

Discussion

Wernicke Encephalopathy (WE) is a rare neurological disorder, potentially fatal but preventable, due to thiamine deficiency. Its clinical presentation may vary among patients not labelled as being at risk, leading to its underdiagnosis [1]. Only 15% of WE are not diagnosed with post-mortem [2]. Thiamine Deficiency can complicate alcoholism, prolonged parenteral nutrition without B1 supplementation, anorexia and any pathology with a nutritional deficiency such as hyperemesis gravidarum (HG), intestinal obstruction, bariatric surgery, hemodialysis or malignant diseases and chemotherapy [3]. In our case, WE occurred after incoercible vomiting due to HG associated with poor intake.

Nausea and Vomiting are very common during pregnancy, especially during the first trimester affecting up to 80% of pregnant women [4,5]. HG complicates 3% of all pregnancies leading to weight loss, volume depletion resulting in ketonuria and or/ ketonemia [6,7]. It occurs at 14-16 weeks' gestation, following more than three weeks of vomiting [8]. As it is, in our case, HG occurred at 12th weeks' gestation after uncontrollable vomiting lasting for two months [9,10].

Thiamine Pyrophosphate, the active metabolite of thiamine, has an important role in the pentose phosphate pathway, key to cerebral energy homeostasis. As a matter of fact, it's an essential cofactor for 3 key-enzymes: alpha-ketoglutarate dehydrogenase complex, the pyruvate dehydrogenase complex and transketolase. Thiamine is predominantly found in the brain. Therefore, its deficiency affects more profoundly in tissues with high thiamine turnover such as neural parenchyma [11-13]. Thiamine is an essential water-soluble vitamin for which the recommended daily allowance (RDA) is 0.4mg/1000Kcal [8]. Among pregnant women, RDA increases to 1.5mg/day [9,10].

In Hyperemesis Gravidarum (HG), thiamine storage rapidly depletes leading to WE [8]. Although many theories have been published, the exact physiopathology is still blurry. Early diagnosis is exacting as the picture presentation is vague and non-specific such as fatigue, irritability frequent headaches and abdominal discomfort [14]. Diagnosis relies on the clinical triad of WE associating encephalopathy, oculomotor abnormalities and gait ataxia [15]. Typical presentation is very rare and may only occur in 10% of all cases [16].

Cognitive changes range from apathy and mild neurocognitive symptoms to behavioral disturbances mimicking an acute psychotic disorder to coma and death [16-18]. Almost a third of patients present with ocular abnormalities such as ophthalmoplegia and specifically horizontal nystagmus [19]. Gait ataxia is found in 23% of the patients. In addition to the WE triad, many findings have been reported: Hypothermia, hypotension, tachycardia, epileptic seizures, hearing loss, absence of deep-tendon reflexes [20]. Approximately 80% of untreated patients with WE develop Korsakoff syndrome associating permanent memory impairment and confabulation [21]. In our case, clinical picture was typical consisting of gait ataxia (kinetic and static cerebellum disorder and disorientation), ocular abnormalities (unequal pupils, horizontal nystagmus and retinal hemorrhage) and neuro-cognitive symptoms (apathy and memory loss). Although WE diagnosis is based on clinical findings, thiamine biological assay may be effective in identifying low plasmatic thiamine in patients at risk of developing WE and in ambiguous presentation [21].

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Imaging investigations rely on Magnetic Resonance Imaging (MRI) as it is the diagnostic tool of choice and not on Computed Tomography (CT) with a 93% specificity and 53% sensitivity [22,23]. MRI findings are wide-spread and vary with severity and duration. Areas of increased T2 and fluid-attenuated inversion recovery signals, decreased T1 signal, and diffusion abnormality surrounding the aqueduct and third ventricle and within the medial thalamus, dorsal medulla, tectal plate, and mammillary bodies can be typically identified on the MRI [21]. In our case, we were unable to order an MRI as it is not available in the emergency department. CT, however, was available and normal. As treatment with thiamine does not have any known side-effects, empirical treatment with thiamine in the form of multivitamins is usually the norm and no diagnostic tool (imaging and biology) should delay treatment [24].

Prompt treatment is key to reverse WE evolution. According to the European Federation of Neurological Societies and the Royal College of Physicians, 500mg of parenteral thiamine should be given 3 times daily until acute symptoms of WE resolve [25]. That was the same protocol followed by our department. In the current Guidelines, in order to prevent WE occurrence in HG, parturient with heavy vomiting and nausea should be administered 100mg of intravenous or intramuscular prophylactic thiamine [26]. As a matter of fact, pregnant women with HG, complicated by WE have not received prophylactic thiamine based on the recommended guidelines [26]. Moreover, Magnesium level should be checked and administered if low because it acts as Thiamine co-factor [21].

Conclusion

WE is rare preventable neuropsychiatric condition due to a thiamine deficiency that can occur in alcoholism and disorders causing malnutrition and malabsorption. HG may induce a B1 deficiency during pregnancy leading to WE. Prevention by prophylactic thiamine in HG is key to avoid the onset of WC. Prompt therapy without waiting for imagining or biological investigations shapes the prognosis. Late or under-treatment steer irreversible complications such as Korsakoff's syndrome [27].

Ethical Approval

Ethics approval has been obtained to proceed with the current study.

Consent

Written informed consent was obtained from the patient for publication of this case report and any accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

Author Contribution

NC: study concept and design, data collection, data analysis and interpretation, writing the paper. HL: study concept and design, data collection, data analysis and interpretation, writing the paper. AS: study concept, data collection, data analysis, writing the paper. NC: study design, data collection, data interpretation, writing the paper. HL: study concept, data collection, data analysis, writing the paper. AS: study concept, data collection, data analysis, writing the paper.

Guarantor of Submission

The corresponding author is the guarantor of submission.

Availability of Data and Materials

Supporting material is available if further analysis is needed.

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